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VOL. II JANUARY 1946 No. 2

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METASTATIC OSTEOMYELITIS FOLLOWING MASTOIDITIS

Case Report No. 41

Dr. George Spence W. H. W. 457069

A four year old colored male was first treated in the out-patient department at Gallinger Hospital, Washington, D. C. for a purulent left otitis media. No specific medication was given and the mother was merely instructed to syringe the ear with peroxide. The purulent discharge cleared up temporarily, but recurred at about monthly intervals. In July 1945, the discharge would not clear up with the peroxide irrigations and the patient was placed on a regime of sulfadiazine without benefit. The patient's general condition was satisfactory and hospitalization was not deemed necessary during this period.

He was first seen at The Children's Hospital on September 13, 1945, and did not seem acutely ill. The abdomen was moderately distended. There was a purulent profuse drainage from the left external auditory meatus, slight tenderness over the left mastoid process and an enlarged lymph gland behind the left ear. X-ray of the mastoid processes at this time revealed a haziness over the left mastoid cells. Sulfadiazine was given and the temperature dropped to normal from September 15th to September 20th. At this time a daily elevation of temperature with usual limits of between 100° and 102° F. was noted. The left ear drum could not be seen due to a polypoid mass occluding the canal. This mass was removed and following removal, the temperature fluctuated even more widely each day. On October 6, 1945, a left mastoidectomy was performed by Dr. Birch and pus was found in the cells, especially at the root of the zygoma. The dural plate was described as being sound at this time.

On October 11th, a sero-bloody discharge was noted coming from the left auditory meatus and a few stitch abscesses developed in the incision of the mastoidectomy. These latter were treated and the healing of the incision continued to satisfactory completion. The ear drainage was found to contain hemolytic staphylococcus aureus. In spite of penicillin and sulfadiazine medication, the daily septic-like temperature continued.

On the 21st of October, the patient was described as appearing non-toxic in spite of continued fever which observation was repeatedly made throughout the further course of the illness. A positive tuberculin at this time, led to the consideration of tuberculosis as the etiologic factor of the continuing fever, but chest X-rays and stomach washings were all negative for acid-fast bacilli.

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At the end of October, 1,720,000 units of penicillin had been given and caused no apparent change in the febrile course or general condition of the patient.

On November 3rd, a mass was noted in the left ramus of the mandible, which was hard and the size of a hazelnut. In succeeding days, it became slightly larger. X-ray of this mass showed it to be a benign bone tumor, probably an osteoma.

On November 13th, the left leg was noted to be favored while walking. X-rays of the femora and pelvis were negative. Spinal taps had been performed on several occasions during preceeding weeks and all spinal fluids had been normal.

On the 19th of November, the patient's temperature climbed to 105° F. and he appeared more toxic. Chest X-rays were again negative. Eye ground examination showed normal fundi. Penicillin therapy was reinstituted and temperature dropped by crisis to normal. However, in twenty-four hours it again began fluctuating daily between 103° and 99° F. The left ear continued to drain during this entire period, but cultures after October, showed only non-pathogenic organisms. Repeated blood cultures taken throughout the course of the illness were negative.

The patient was seen in early December by Dr. Lambros from the Neurological Service and he made a presumptive diagnosis of cerebellar abscess secondary to mastoiditis.

A subsequent examination in the second week of January, disclosed more localizing signs. There was a left lateral nystagmus, left muscular hypotonia, left ataxia and incoordination and a positive Romberg. The child walked on a wide base and swung the left leg in an arc when walking. Atrophic changes in the musculature of the left leg were noted, the right thigh measuring 3 inches more in diameter at mid-thigh than the left.

An air-encephalogram was performed and disclosed a moderate degree of hydrocephalus with upward displacement of the posterior horn of the left lateral ventricle. A mass occupied the middle and upper portion of the fourth ventricle which Dr. Lambros believed to be a cerebellar abscess. There were multiple metastatic lesions in the calvarium, many of which appeared to be healing. A second area of metastatic osteomyelitis was discovered in the left fibula.

On the 24th of January, a craniotomy was performed. The cerebellar tonsil on the left was herniated through the foramen about one centimeter. Dr. Lambros stated, on probing, that he found an encapsulated mass about 1.5 cm. beneath the surface about 1.5 cm. in diameter. The petrous pyramid was soft, yellowish and necrotic and adjacent dura was congested. The mastoid bone showed abundant granulation tissue. There was no attempt made to remove the cerebellar mass.

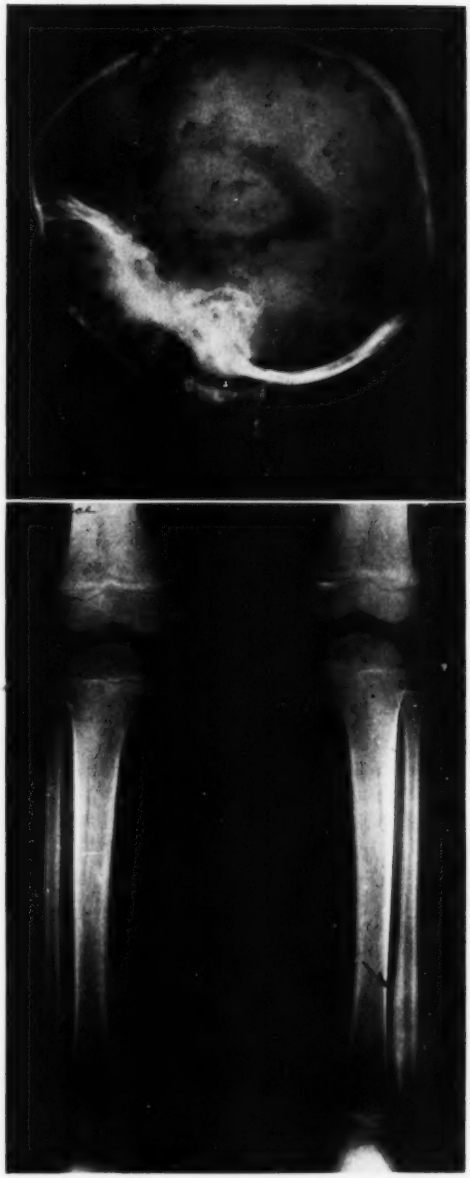

FIG. 1. (Upper) NOTE MULTIPLE METASTATIC AREAS OF OSTEOMYLITIS IN THE CALVARIUM

FIG. 2. (Lower) OSTEOSCLEROSING METASTATIC LESION IN THE FIBULA

The post-operative condition of the patient continues to be good after forty-eight hours. There has been a moderate amount of vomiting. The patient is conscious and mentally alert at this time.

Summary

A four-year old colored boy, admitted in October to The Children's Hospital, was found to have a chronically draining left ear of eight months' duration. A mastoidectomy was performed and the diagnosis of acute left mastoiditis confirmed. The illness was marked by a prolonged febrile course, which was little affected by the use of sulfadiazine and penicillin. The signs of ataxia and incoordination on the left side led to the neurologist's diagnosis of left cerebellar abscess secondary to acute left mastoiditis. Encephalograms confirmed the suspicion of a cerebellar tumor or mass and at operation, Dr. Lambros states, that he found a mass located in the left cerebellar hemisphere, as well as an extensive necrosis of the left petrous pyramid. Other metastatic osteomyelitic lesions were disclosed by X-ray to be located in the left fibula and in the calvarium. The cerebellar mass was not removed. The post-operative condition of the patient remains good.

Discussion

Direct extension of infection into and beyond the temporal bone from an otitis media and mastoiditis is the most common source of cranial osteomyelitis in children. Involvement of the frontal bone from a frontal sinusitis occurs occasionally. Osteomyelitis may result from any bacteremia. It may also be caused by direct extension from infections of the scalp and head wounds. The most common infecting organism is the Staphylococcus aureus. Osteomyelitis of the bones of the skull differs from that seen in other bones in almost never being a primary infection. This is due to the absence of a vascular epiphysis and metaphysis which are so frequently the seat of infection in the long bones.

The subperiosteal osteomyelitis noted in the fibula in this case is most frequently associated with an extensive medullary type of osteomyelitis as a metastatic lesion. The pyogenic organisms which produce the original medullary involvement, circulating in the blood stream, find lodgment in some bone by way of the subperiosteal blood supply and even slight trauma such as the pressure of bedclothing may be a factor in its development. The confinement of the infection to the periosteum leads to layer after layer of new periosteal bone being laid down. This layer-like appearance may occasionally cause some difficulty in differentiating its X-ray appearance from that of a Ewing's tumor.

SPECIAL REPORT

ATELECTASIS IN INFANCY AND CHILDHOOD

Dr. William Anderson

Pulmonary atelectasis is an airless condition of a lung or part of a lung—the word being derived from the Greek words "ateles" meaning incomlete, and "ektasis" or extension. The perfect state of this condition exists in the fetus in utero—which fits this derivation accurately. In later life, however, the term applies to a condition of collapse of previously air-filled tissue.

The initial expansion of the atelectatic lungs of the new-born infant is thought to be due to the stimulation of the respiratory center by the rising CO_2 content of the blood stream of the infant after the cord is tied. Van Reuss has shown that the lung capacity is about 17 cc. 20 minutes after birth and this is doubled from $3\frac{1}{2}$ to 6 hours after birth. It is definitely established that the lungs do not completely expand for several days even under normal conditions.

There are some investigators, however, who believe that respiration is not initiated at birth, but extends back into embryonic life—Snyder and Rosenfeld in Baltimore and Bonar and Fenning in Salt Lake City have been instrumental in renewing interest in intra-uterine respiratory movements. Interest in this subject dates back to the sixteenth century and in 1781 Winslow observed in the fetuses of dogs and cats, in which the umbilical cords had not been disturbed, rhythmic contraction and dilatation of the nasal orifices, expansion and contraction of the thoracic wall and movement of the fetal abdomen in the amniotic fluid after the uterus had been exposed. The investigators mentioned studied these movements by motion pictures and also by kymographic tracings, recording fetal respiratory movements, maternal respiration and pulse. Bonar describes the fetal respiratory act as resembling that of the new-born animal with the chest being raised suddenly with abdomen retracted or vice versa, with ribs moving up and down, and in most instances the movement was accompanied by opening and closing of the mouth and dilatation of the nostril.

These observations have considerable bearing on atelectasis of the newborn, since it was observed how marked was the depressant action of anoxemia and narcosis on the sensitive fetal respiratory system. It seems to me that this opens a channel for further investigation in obstetrical anaesthesia and analgesia. To quote Snyder and Rosenfeld, "Caution

in the choice of anaesthetics should be more satisfactory in preventing depression of the respiratory function than later attempts in resuscitation."

An interesting observation on the respiratory physiology of the newly-born presents itself when one considers that at birth the respiratory tree is probably completely filled with amniotic fluid. When turned upside down the pharyngeal mucus is manually removed. Mouth or electric suction is used if necessary. At the first inspiration of the infant a great deal of the amniotic fluid is aspirated. Farber and Sweet studied 124 infants who had lived from 2 hours to 5 weeks, and demonstrated material from the amniotic sac in the lungs of 88%. They attributed these findings to aspiration of amniotic fluid during intra-uterine asphyxia and were inclined to agree with Reifferscheid that the glottis of the fetus in utero was closed. Snyder and Rosenfeld support the opposite view that inspiration of the amniotic fluid is a normal function of fetal respiration, with a tidal flow, affording a mechanism for the dilatation of the future air passages. They injected india ink into the amniotic sac of rabbit, cat and guinea pig fetuses and waited a few minutes before clamping off the neck of the fetus. In fetuses which had showed respiratory movements the lungs were blackened, while in the quiescent fetuses, such was not the case. Certainly it is no small wonder that more cases of atelectasis are not observed at the new-born period.

Atelectasis of the newly-born is of course more common among prematures or infants suffering from congenital debility. Bodily weakness and failure of the respiratory center both play a part. Intracranial hemorrhage or edema, fractured skull, or congenital cerebral defects are frequently accompanied by atelectasis. In premature infants Wilson and Farber feel that part of the lung is unexpendable and that the atelectasis is partly due to the cohesion of the moist surfaces which form an obstacle to the entrance of air. Physical findings are those of cyanosis, rapid shallow respirations or occasionally no tendency to breathe at all. It is sometimes difficult to outline an area of dullness or bronchial breathing. Probably the most common finding is the fine, velvety crackling roles heard posteriorly at the lung bases near the spine. X-ray is of course of value in diagnosis if feasible.

Treatment consists of an oxygen tent constantly and intermittent inhalations of a combination of oxygen and CO_2. Yandell Henderson considers 7% CO_2 as optimal. Artificial respirations should be undertaken with caution. The Drinker respirator in my experience has been valueless in full term infants and contra-indicated in prematures. The Flagg tracheal insufflator is of value in experienced hands. The Kreiselman apparatus is simple, harmless and fairly helpful.

After the new-born period atelectasis may be divided into two types: (1) Pneumothoracic with air admitted to the pleural cavity and (2) obstructive. It is my purpose to discuss these two types briefly and present a case illustrating an unusual example of each of the two types.

Pneumothoracic atelectasis occurs in the presence of a variety of conditions in which air is admitted to the pleural cavity. The important etiological factors are infection and trauma. Tuberculosis plays a very minor role in infancy and childhood, whereas in adults it is considered the etiology in 80 to 90%. In 84 children with pneumothorax: pneumonia 36%; gangrene of lung 13%; emphysema 13%; pertussis 5%; lung abscess 4% bronchiectasis 4%; foreign body 4%; congenital defects 5%; typhoid, influenza and operative procedures 16%. The rupture of a congenital lung cyst is occasionally reported, as is the rupture of an emphysematous pulmonary bleb. Spontanecus pneumothorax and pyopneumothorax are of rather frequent occurrence. Traumatic pneumothorax is caused by crushing or piercing wounds or following lung aspirations, or other surgical procedures.

Physical findings in the *Pneumothoracic type of atelectasis* are (1) diminished respiratory excursion on the affected side and increase on the opposite side; (2) intercostal spaces are leveled and sometimes bulging; (3) tympanitic percussion note; (4) absent breath sounds; (5) shifting of the heart and mediastinum *away* from the affected side; (6) cyanosis, restlessness, dyspnea, rapid thready pulse and collapse. Treatment naturally varies with the underlying cause and time does not permit a detailed discussion of this phase.

For completeness it might be wise to mention Hydothorax, Hemothorax and Chylothorax—producing atelectasis and not fitting into the Pneumothoracic or obstructive types.

The obstructive type of atelectasis without pneumothorax is associated with a variety of causes. First of all it is well to visualize according to Chevalier Jackson—"The entire thorax as a single cavity with a resilient air-tight curtain dividing it into two cavities, each occupied by an air bag, the right and left lung respectively. When the air enters each bag equally the atmospheric pressure distends each bag equally; when entrance is obstructed on one side a differential is created diminishing the bulk of that bag as compared to its fellow on the other side of the curtain. To take up the diminution of bulk the dividing curtain moves from its central position over toward the side of diminished bulk."

Four types of bronchial obstruction are explained by the expansile check-valve as described by Chevalier Jackson:

(1) Bypass valve obstruction with air passing both ways in diminished volume and producing a wheeze.

(2) Check valve permitting air to enter but not escape, producing emphysema.

(3) Reversing the check valve permitting air to escape but not to enter, producing a rapidly developing atelectasis.

(4) Stop valve obstruction allowing no air to enter or escape, causing a slowly developing atelectasis by the gradual absorption of the retained air by the circulation.

Conditions producing valvular obstruction are (1) Foreign bodies, either opaque or non-opaque or endogenous substances such as secretions, pus granulations or tumors acting as such; (2) Malignant growths in endobronchial, murobronchial and peribronchial forms most commonly Lymphosarcoma or Hodgkins Disease; (3) Adenopathy—especially with malignant and tuberculous conditions; (4) Congenital anomalies of both the check-valve and stop-valve varieties; (5) Inflammatory mucosal swelling which are extremely common in children; (6) Granulations and granulomata preceded by inflammation; (7) Bronchiectasis due to viscid secretions; (8) Secretions when hardened into crusts or membranes favored by (a) increased fibrin due to excess of fibrinogen, secondary to inflammation or passive congestion, (b) absence of cough reflex and resultant stagnation, (c) administration of morphine and atrophine, (d) character of bacterial infection. Under this type of instruction Post operative atelectasis can probably be classified. (9) Asthma; (10) Diphtheria; (11) Blood clots—secondary to new growths, tuberculosis, foreign bodies, spirochetosis, pneumonia, inflammatory diseases and operations on tonsils, tongue and sinuses; (12) Tuberculosis—endobronchial as tuberculoma; murobronchial with lesion in bronchial wall; and peribronchial as adenopathy.

Diagnosis of atelectasis and emphysema is readily made by physical signs and X-ray or fluoroscopic examination. The character of the obstruction can be determined only by bronchoscope.

Physical findings will vary considerably with the underlying type of obstruction and the X-ray and fluoroscope are very important in the diagnosis. It is well to remember that in obstructive atelectasis the shift of the mediastinum is *towards* the obstructed side, whereas in emphysema or pneumothoracic types the shift is *away* from the obstructed side. Fluoroscopic examination in obstructive emphysema reveals a sidewise mediastinal shift on expiration, returning to normal position on inspiration; whereas in obstructive atelectasis the mediastinum remains fixed in both phases of respiration.

The character of the obstruction can be determined only by a bronchoscopic examination and most observers feel that this should be done in every case of obstructive atelectasis. "In children this type of atelectasis is so often due to bronchoscopically curable valvular obstruction that the diagnosis of other types of atelectasis should not be made until after diagnostic bronchoscopy has excluded bronchial obstruction," to quote Jackson. The cure of the atelectasis is certainly a preventative of later suppurative bronchitis, bronchiectasis, or lung abscess.

RUPTURE OF THE KIDNEY

Case Report No. 42

Dr. C. J. Tichenor P. W. 46143

P. W., white female, aged 9 years, due perhaps to the lack of caution and the spirit of adventure on January 6, 1946 fell a distance of four feet striking the left renal area against some bricks which were set in cement as a decorative edging. This school aged girl was not particularly perturbed by this accident and proceeded to walk home. At home she complained only of soreness in her left flank. At this time there were no other complaints or symptoms. The family private physician was called and the child was admitted to the Children's Hospital at 4:30 p.m. January 6, 1946. At no time did she lose consciousness.

On admission this child did not appear seriously ill. She was alert and cooperative and complained only of pain in the left hypochondrium. There was a history of trauma as stated and evidence of a small abrasion in the left mid-axillary line over the 6th rib. Compression of the thoracic cage did not elicit the pain of a fractured rib and there was no pain on deep inspiration. There was definite tenderness on palpation over the left side of the abdomen more marked in the left lower quadrant, but also present in the left hypochondrium. This pain was elicited on pressure. Pulse at this time was 128, blood pressure 102/60. The patient seemed thirsty.

Two hours later she expressed a desire to void, this act being performed with difficulty and accompanied by the passage of about 8 c.c. of bright red blood. No evidence of trauma to the external urinary meatus was noted. One hour later the patient succeeded in passing 350 c.c. of grossly bloody urine with several large clots. She felt somewhat relieved at this time although the flank soreness and pressure tenderness in the left hypochondrium persisted. Blood pressure and pulse remained constant. With the history of injury to the flank, localized pain in this region accompanied by tenderness on pressure and gross hematuria, kidney damage was suspected and four hours after admission an intravenous pyelogram was done. Examination of the abdomen by means of a flat plate revealed the presence of a spina bifida occulta of the first sacral segment but no other evidence of abnormality. Examination of the genito-urinary tract after the intravenous injection of dye at the end of 3, 10 and 25 minutes revealed the right kidney, pelvis and calices to be normal in size, shape and position. The left kidney, pelvis and calices were never satisfactorily outlined and the pelvis appeared to be displaced medially, possibly due to hemorrhage.

There was no abnormality of the bladder. Both kidneys were functioning, the left not as well as the right.

Since this appeared to be an isolated kidney injury with no evidence of shock or urine extravasation and no evidence of injury to other viscera was apparent, conservative treatment was maintained and the patient placed on absolute bed rest, local applications of an ice bag to the left

FIG. 1. NOTE DEFORMITY OF THE PELVIS OF THE KIDNEY

renal area and codeine given for sedation. The blood count was 9 gms. of hemoglobin, 2,740,000 erythrocytes, 6,200 leukocytes with a normal differential count. Special emphasis was placed upon careful observation of the patient for bleeding by daily blood counts and observation of the pulse and blood pressure. Hemoglobin on the second day was 7.4 gms. and the erythrocyte count was 2,670,000.

To combat the anemia a transfusion of 200 c.c. of whole blood was given

on the third day and repeated on the fourth day. Blood pressure remained rather constant ranging between 90 and 110 systolic and 50 to 70 diastolic. The temperature fluctuated between 99 and 102° and her hemoglobin at the present time after therapy is 11 grams with erythrocyte count of 3,930,000. Hematuria continued for the ensuing several days and for the three days prior to this presentation urine was still wine red in color. Transient pain and dizzy spells accompanied by headache were complained of for the past two days. Her pulse has dropped from 128 to 90 at the present time and pain is still complained of upon pressure over the entire left flank.

The diagnosis of ruptured kidney has been entertained and the general condition of the patient appears good but she is still under close observation for any evidence of continued bleeding or perirenal infection.

Discussion

Dr. Meredith F. Campbell, Professor of Urology at New York University makes the statement that the kidneys are injured more often than any other urinary organ in children. It is true that the kidneys are fairly well-protected by surrounding bony and muscular structures, but also well known that they may be injured by abdominal, loin, or lumbar blows, by indirect force, by sudden muscular extension or by penetrating wounds. In the normal child approximately a quarter of the kidney lies below the costal margin, and with the inspiratory phase of respiration, as much as half may extend below the last rib. This is particularly true on the right side. Malpositioned or enlarged kidneys are even more susceptible to direct trauma. Paradoxically the spine often acts as the receiving point of kidney crushing when blows are received in the lateral loin or anterior abdomen. Lower costal fractures may inflict contusions or lacerations on the kidneys, particularly if the blow comes from behind. The fact that the right kidney statistically seems more vulnerable may be partially explained by the fact that its normal mobility is restricted somewhat by the relative fixation of its position by the liver, inhibiting its possibilities of sliding out from under a blow. Children of course have a scarcity of perirenal fatty tissue which serves to increase their vulnerability. The school age children (under 15) would seem to suffer the heaviest incidence of renal injury.

Mechanics

The kidney is an encapsulated organ filled normally with blood when subjected to a sudden blow. Hydrostatically the "force exerted upon any part of the fluid is transmitted equally in all directions through the

mass." Changes within the kidney, therefore, are those of a bursting force, the damage ensuing depending upon the severity of that force. The force being mild may result only in minor laceration involving the cortex and capsule, or the medulla and adjacent pelvis. The force being severe may cause the organ to be contused, fragmented or even totally bisected. It is quite possible with a mild force for neither capsule nor pelvis to be fissured.

When the kidney is ruptured the degree and course of urine extravasation or hemorrhage will depend upon the extent and location of the fragmentation. Loose perirenal fat surrounds the organ, so that with cortical or pelvic lacerations, extensive hematoma or extravasation, and even peritoneal laceration may follow. In the case of the latter sequelae there would be blood and/or urine in the abdominal cavity.

Pathology

Renal injuries may be classified as:

(a) Contusions
(b) Lacerations
(c) Ruptures
(d) Crushings
(e) Penetrating wounds

Mild trauma usually results in contusion, manifesting itself by ecclymosis with very small subcapsular tears radiating in many directions from the point of impact. Very often a small subcapsular hematoma develops and blood extravasates into the renal tubules, identifiable either microscopically or macroscopically.

With violence being more severe the kidney may be lacerated or ruptured, the ruptures corresponding more or less to the distribution of the arterial trees. The cortex may be involved, with or without rupture of the capsule, with or without injury to the vessels of the perirenal fat. Rupture of the capsule and cortex alone does not cause urinary extravasation. The tear may extend into the pelvis, or by contre-coup the laceration may be confined to the parietal medulla and adjacent pelvis.

It is when a cortical laceratin establishes a pelvo-perineal path that extensive urine extravasation exists, which may extend down to the pelvis, scrotum or abdominal wall, or up to the subphrenic space, or even if the peritoneum is torn into the abdominal cavity. Under the age of 10 years, the kidney is separated from the peritoneum by an underdeveloped anterior sheath of perirenal fascia, so that tears in the anterior position of the kidney are usually accompanied by tears in the peritoneum—and a poor prognosis.

Symptoms

Kidney injury is manifested by one or more of the following symptoms: shock, renal pain, tenderness in the loin, inspiratory pain, mass in the loin, hematuria, pallor, a falling blood pressure, a diminution of circulating erythrocytes and hemoglobin, a variable elevation of the white cells and occasionally anuria and coma.

Diagnosis

Careful history of the nature and violence of the injury and the location of impact is a prerequisite, to be followed by the immediate post-traumatic reaction. Inspection may reveal the site of external trauma, but a thorough physical examination is still necessary, paying particular attention to the abdomen. It is entirely possible for coexistent injuries of the pleura, diaphragm and peritoneum to be of more importance than the renal injury. Examination before abdominal rigidity occurs is of importance, such data often being unobtainable at a later date. Blood pressure should be recorded and a complete blood count accomplished immediately. Urine should be carefully examined for blood. As soon as possible X-ray study of the urinary tract should be made to determine:

(a) Alterations in the kidney or psoas muscle outlines.
(b) Anomalous condition of the G. U. Tract.
(c) Skeletal fractures.

If the condition of the patient permits, an excretory urographic examination is most helpful in determining the site and extent of the urinary tract damage. A renal pelvis on the uninjured side appearing normal gives the confidence necessary for either radical or conservative treatment. With bleeding into the pelvis the pyelogram may show pelvic irregularity, filling defects or complete obliteration. Laceration of the pelvis will usually be shown by intrarenal or perirenal extravasation of the dye media.

The gravity of the situation, however, is indicated by repeated blood counts and blood pressures. Thus a rapidly falling erythrocyte count and hemoglobin calls for large transfusions and immediate surgical intervention. When exploration can be justifiably delayed however, more thorough urological examinations should be undertaken. In general in renal injury, retrograde pyelography is contra-indicated because it may incite fresh bleeding or introduce infection. Excretory urography is the diagnostic method of choice, especially in the less severe appearing injuries. The patient should not be allowed to die of shock or hemorrhage while attempt is being made to make an accurate and inclusive anatomical diagnosis.

Differential Diagnosis

The site of external trauma is not prima facie evidence of the exact organ that may be injured, nor does it tell us the extent of damage. When hematuria is profuse or where a mass in the loin quickly follows a history of lumbar injury and is accompanied by a falling blood count, kidney injury is the likely diagnosis. Nevertheless associated intra-abdominal traumatic lesions often occur, and in the acutely ill patient in shock, can rarely be diagnosed except by exploration. Intra-peritoneal hemorrhage shows itself by a falling erythrocyte count and high leucocytosis, along with shock, free abdominal fluid, tympanites, abdominal rigidity and tenderness. However, shock, rigidity and tenderness are also manifestations of renal trauma and of intra-peritoneal injury without hemorrhage. Abdominal exploration may be the only means of revealing the true condition.

Treatment

Most children with renal injury are not in profound shock initially, but many gradually develop this condition, which may be fatal. Unless there are definite signs of marked and continuous bleeding, supportive measures and the treatment of shock should be conservatively used. External heat is initiated, pain is controlled by morphine and the body fluids are restored by whole blood transfusion and/or intravenous infusion of 5% glucose in normal saline. Thyroid extract intra-muscularly may increase renal excretion. Gentle examination of the loin and abdomen should be performed for the appearance or increase in size of a mass. Blood counts should be repeated every half hour. As soon as the condition of the patient permits, flat plate of the abdomen and excretory urography should be accomplished. Cystoscopic examination should only be done as a last resort when other diagnostic means fail.

When hematuria and bleeding is not alarming and shock is mild or absent conservative methods would seem best. The patient should be kept quiet until there has been no hematuria for at least one week. Continued bleeding, the reoccurrence of hematuria, perirenal extravasation, suppuration or anuria of twenty-four hours duration, point to conservative treatment being inadequate and make surgical intervention advisable. Meredith Campbell states that about two thirds of all patients with renal trauma may be successfully treated conservatively and that their relative superior condition usually justifies cautious urologic examination, including early excretory urography.

Operative intervention is urgently indicated for: the control of bleeding in severe hemorrhage, persistent anuria, intra-peritoneal trauma and the drainage of infection following injury. Nephrectomy should be delayed until palpation and intravenous pyelography or cystoscopy have dis-

closed a good kidney on the other side. More often nephrectomy should not be done when conservative surgical treatment can be substituted.

Complications of Renal Injury

Apparent spontaneous healing of an injured kidney may be followed by secondary hemorrhage, especially where conservative surgical treatment has been employed. These may not occur until seven to ten days post injury. Secondary suppurative lesions such as renal, peri-renal or subphrenic abscess are often fatal. Deformities and obstructive post traumatic sclerosis may cause hydronephrosis.

Prognosis

The prognosis depends upon the severity of the renal injury and its associated complications. Michelson reported three deaths in 30 cases of renal injury conservatively treated and these occurred from severe complicating injuries. Intelligent treatment is usually conservative but in certain instances must be surgical, prompt and radical. Death most often results from intra-peritoneal or pulmonary complications.

CLINICO-PATHOLOGICAL CONFERENCE

Directed by Dr. E. Clarence Rice

Assisted by Dr. Robert B. Sullivan

Case Report No. 43

Dr. Robert Sullivan

A three month old colored female was admitted to the Children's Hospital on January 23, 1940 because of jaundice of two weeks' duration. During this period the stools were said to be "clay-colored" and the infant vomited a small amount after each feeding every day. The vomiting was not forcible. Her appetite was good and she had seemed healthy until the onset of the jaundice which rapidly became marked.

She was born at home by a spontaneous normal delivery and the birth weight was six pounds. There was no breast feeding and the formula was six ounces of evaporated milk with eighteen ounces of water plus one and one half tablespoons of karo. No cod liver oil was administered but she received three ounces of orange juice daily. Her development had been normal. A serological test for syphilis had not been done and her only previous illnesses were "occasional mild colds."

The father was well. Her mother had a four plus Wassermann during this infant's pregnancy and she received no anti-luetic therapy. She was said to have Hutchinson's teeth. There were two living siblings about whom little is known. Anteceding these children the mother bore a premature stillbirth at eight months.

The patient was fairly well developed, poorly nourished, moderately ill and somewhat dehydrated. The sclerae and skin were markedly jaundiced. The rectal temperature was 99.2°. Cervical, epitrochlear, and inguinal nodes were palpable. A small umbilical hernia was present. There was hepatosplenomegaly with the liver extending three centimeters and the spleen one centimeter below the costal margin. No other positive findings were elicited.

The hemogram, urinalysis, coagulation time and bleeding time were normal. On admission the prothrombin time was 100% but in mid-course another estimate was 61%. Two Kahns were negative as was the spinal fluid Wassermann. A routine examination of the spinal fluid revealed it to be normal and the colloidal gold curve was 00001221000. The red blood cell fragility was moderately decreased. There was no sickling of the red cells. Tests for the presence of bile pigments revealed the Van den Bergh to be prompt and biphasic and the icterus index to be forty

units. The urine was reported negative for bilirubin and stool negative for urobilin. These tests were not repeated. An X-ray study of the long bones revealed them to be normally developed with no evidence of lues. A few days before the infant's death an X-ray of the chest showed an area of opacity in the right apex.

During the first two weeks the temperature range was from 99° to 104° and during the last five weeks it was from 98° to 102°. There was a slow weight gain from 8 pounds 1 ounce on admission to 8 pounds 12 ounces at death. A mild diarrhea was easily controlled in the first week. Thereafter the stools numbered 3 to 4 daily and were light yellow or light cream-colored. Vomiting was minimal. The liver became larger and its edge extended to the umbilicus whereas the spleen receeded and was not palpable after the first week. Although the jaundice remained about the same, the infant's condition in general gradually declined. There was a thin nasal discharge and a cough during the 2nd week which was followed by drainage of yellow pus from the right ear for two weeks. This in turn was followed by drainage from the left ear during the last two weeks. In the last week there were rales in the left chest and signs of consolidation over the right chest. Death occurred on the forty-eighth day of hospitalization.

Differential Diagnosis

Dr. John M. Elliott: We are presented here with a case which in general seems to have an insidious onset and appears to become progressively worse and in the differential diagnosis this fact becomes important.

The first consideration is that of obstructive lesions to the biliary system. It is true that congenital atresias show symptoms before this age, however, I am thinking of a partially occluded or stenosed passage which may be adequate for biliary drainage until a small mass of inspissated bile or some other similar material would lodge in the narrowed passage and thereby produce obstruction. The fact that the stools were acholic during the entire course of the illness seems to point very definitely toward an obstructive lesion or some lesion which makes the excretion of bile deficient or impossible.

Catarrhal jaundice is not common in this age group and is more often seen in school aged children. However, there are several cases of this condition reported in infants. There is generally a more acute onset, the temperature is high from the start, the systemic toxic reaction is more extensive than is apparent here and the jaundice begins soon after the onset. In the case today the jaundice appeared to antedate any systemic reaction. It appears to me that the child was initially jaundiced and later became sick. Nausea and vomiting and the biphasic Van den Bergh are common to catarrhal jaundice and both findings are present in this case.

Cirrhosis, particularly of the type described by Hanot, is not commonly seen in this age group but in any differential diagnosis of jaundice it merits consideration. In certain parts of the world, notably in India, cirrhosis is one of the common causes of infant mortality. Chronic biliary obstruction which may or may not be clinically apparent will produce cirrhosis in children and in an amazingly short time. With a history of jaundice, fever and mild gastro-intestinal upsets from no apparent cause, cirrhosis must be ruled out.

I would like to question at this time the veracity of the negative test for bile in the urine. It would seem that any degree of icterus sufficient to be clinically apparent as well as cause persistently alcoholic stools would cause biliary pigments to be found in the urine.

The next consideration is that of liver abscess and this seems to be a good bet. Hepatic abscess may be quiescent or at least not apparent in the infant until it has reached sufficient size and has done damage enough to cause jaundice. Gastro-intestinal symptoms are common findings. A fever of the nature we are presented with here is what one might expect. The liver became larger, the child became more toxic until finally suppurative middle ear infections occurred and what can be assumed to be a terminal pneumonia was the cause of death. An original liver abscess could well be the mother site for such a widespread septic course.

Liver cysts especially those of congenital origin deserve only passing consideration. However, they begin to show in this age group and can very well present such a picture. Pulmonary cysts frequently accompany hepatic cysts and could explain the roentgen interpretation of the chest.

Sepsis is an important cause of jaundice in children of any age, especially in infants. Again the initial picture does not seem to be severe enough to be caused by a septicemic process. Sepsis may well have developed during the course but I don't believe it was the initial etiologic process.

Tuberculosis of the liver or the biliary system must be mentioned. Tuberculosis can explain any clinical picture and could well explain this one.

The baby certainly has a good background for a syphilitic infection. We are not told if the previous two children were syphilitic which well they may be. The history of a previous late abortion is characteristic of a luetic pregnancy. The serology in this case seems to be conclusively negative. I would like to ask someone here that presuming a child was born of a syphilitic mother at what time are negative serological reactions considered reliable in the infant? The authorities that I have read claim that from eight to twelve weeks of life should be the upper limit. Jaundice occurs in congenital lues in less than 2% and the stigmata of congenital lues are not apparent in the protocol, nevertheless, I believe that this diagnosis merits serious consideration.

The lipoid storage diseases I will quickly pass over. Gaucher's and Neiman-Pick's appear a few months after birth. Anemia, hepatosplenomegaly and lymphadenopathy are usually present and cholesterolemia is characteristic of these diseases.

In considering the anemias one can only wish we had more hematological findings to work with. Repeated red blood counts and morphological studies would have been helpful. Sickle cell anemia has its onset in older ages characteristically and is rare at this age. The onset is usually in the form of a hemolytic crisis which doesn't seem to be the case here.

A resistance to the fragility test is characteristic of this disease and occurred in this case. Therefore the one negative sickling test should have been repeated. A massive hemolysis of sickle cells could have occluded the liver radicals and thus have caused the evidence of obstruction which we have here and cause a diphasic Van den Bergh.

Congenital hemolytic jaundice is rare at this age and the fragility test if accurate would seem to rule it out. A hemolytic anemia of the type described by Von Jaksch is most frequently seen between the ages of 6 months to 2 years and is relatively infrequent in this country. A decreased white cell count and a relative lymphocytosis as did occur in this case, although not marked, do also occur in Von Jaksch's pseudo-leukemic type of anemia. This condition has a multiplicity of causes many of which could have applied to this case. Large liver, spleen and palpable lymph nodes as well as jaundice and fever are eventually seen in this anemia which is frequently fatal.

My first diagnosis in this case would be that of hepatic abscess with a secondary septicemia, multiple infection in the ears, and a terminal pneumonia.

Secondly I would favor some obstructive lesion either in the liver or the excretory duct system and lastly congenital syphilis.

Pathological Diagnoses

1. Bilateral broncho-pneumonia.
2. Cirrhosis of the liver.
3. Absence of the common bile duct.

Dr. Robert B. Sullivan: At autopsy the findings of interest were in the lungs and liver. The lungs were heavy and of a mottled dusky red color with sectioned surfaces revealing irregular areas of congestion and consolidation bilaterally. Microscopic examination of the tissue confirmed the impression of congestion and bronchopneumonia. The liver was almost twice its normal weight and extended five centimeters below the costal margin. Its external surface was mottled gray and greenish gray-brown. The tissue sectioned with increased resistance disclosing promi-

nent fibrous trabeculae and an olive-brown parenchyma. Although the gall bladder, biliary and hepatic ducts were present, careful dissection revealed no evidence of the common bile duct. Microscopic examination of the liver tissue demonstrated marked hyperplasia of the connective tissue and the bile ducts with corresponding encroachment of the parenchyma. There was considerable congestion and slight leucocytic infiltration. The deposition of biliary pigment was surprisingly small in amount. In Levaditi stained sections no spirochaetes were seen.

On reviewing this case one is struck with the late appearance of jaundice (during the tenth week). In the reported cases of absence of the common bile duct jaundice has appeared during the first month. Perhaps the jaundice was unrecognized but present earlier in this colored infant. The sclerae are not seen easily in infants or perhaps the common duct was present at birth and later atrophied. This is not likely since it is believed now that the etiology of the bile duct atresia lies in failure of canalization during development rather than due to mechanical blockage or infection processes. In this case the negative serological tests, the negative X-rays and the absence of gross or microscopic evidence of lues makes this diagnosis extremely unlikely. The diagnosis of bile duct atresia can be suspected in infants with intense jaundice constantly clay colored or white stools and a distended abdomen which persists for one month and cannot be otherwise explained. About twenty per cent of these infants can be benefited by anastomosing operations.

ANNOUNCEMENT

GEORGE WASHINGTON UNIVERSITY

Postgraduate Course in Pediatrics

Four weeks of instruction are offered, beginning March 4, 1946. This is divided into two periods of two weeks each and the physician may elect one or both periods. The instruction is full-time and intensive throughout.

The first two weeks of instruction is largely didactic and is presented at the School of Medicine. Among the topics considered during this period are the following: "Physiology of the Newborn"; "Basic Food Requirements"; "Metabolic Diseases of Childhood"; "Behavior Problems"; "Immunizations"; "Parent-Infant Relationships"; "Epidemic Diarrhoea of the Newborn"; "Examination of the Well Baby"; "Feeding of the Older Child"; "Care of the Premature"; "Symposium on Rheumatic Fever"; "Epidemiology of Streptococcus Infections"; "Symposium on the Antibiotics"; "Symposium on Poliomyelitis"; "Clinic; Nephritis in Childhood"; "Clinic; Congenital Syphilis"; "Clinic; Childhood Tuberculosis." Tuition for this two weeks' period is $50.00.

The second two weeks period will be presented almost entirely in the hospital. Opportunity will be afforded to examine patients and informal discussions will be encouraged. Registration during this period is limited to fifteen. Among the topics considered during this period are: "Studies in Penicillin Absorption and Excretion"; "Isolation Technique"; "Diagnostic Procedures Utilized in Infectious Diseases"; "X-ray Conference—Pulmonary Infections"; "Factors in the Reduction of Fetal and Infant Mortality"; "Causes of Prematurity"; "Fetal Resuscitation"; "Technical Procedures in Pediatrics"; "Difficult Feeding Problems"; "Prevention of Behavior Problems in Childhood"; "Coeliac Syndrome"; "Eye Problems in Children"; "Orthopedic Problems in Childhood"; "Pediatric Neurology", etc. Tuition for the second two weeks' period is $100.00.

For these courses, the faculty of the School of Medicine, which numbers more than two hundred, will be supplemented by prominent guest physicians and surgeons from other institutions. More than 175 prominent guest scientists will appear in the schedules of the various courses, Dean Bloedorn stated. These include representatives from the faculties of more than twenty Medical Schools and Universities, including the following: University of Chicago, Columbia University, Cornell University, Duke University, Georgetown University, Harvard University, Jefferson Medical College, Johns Hopkins University, University of Maryland, New

York University, University of Pennsylvania, Southwestern Medical College, Stanford University, Temple University, University of Tennessee, Tufts College, Tulane University, Vanderbilt University, University of Virginia, Medical College of Virginia, Wayne University, University of Wisconsin and Yale University.

When applying for a course of instruction, the physician, veteran or otherwise, must submit his check or money order for $25.00. This will be refunded in full when actual registration is completed at the beginning of the course. Checks should be made payable to "The George Washington University" and forwarded to the undersigned.

It is difficult to secure housing in Washington at this time. Wherever possible, it is suggested that the veteran make his own arrangements. It is hoped that a limited number of rooms and apartments can be made available, however. If you desire aid in this connection, please state your requirements clearly in a letter to this office.

If the need is apparent, it is hoped that the material to be presented in these courses can be bound into volumes. This should make a very convenient reference book with each chapter up-to-date. Before this is undertaken it is necessary to learn what the demand will be. If interested, please mark your application form accordingly. They will be sold at cost to those registered in the course. The cost of these books will not be covered by the G. I. Bill of Rights.

Information for Veterans

The courses of instruction described on the enclosed sheets are designed primarily to meet the needs of returning medical officers. The full cost of these courses can be covered by the provisions of the G. I. Bill of Rights.

In order to be eligible for the educational provisions of this Bill, the veteran must present his "Certificate of Eligibility and Entitlement" at the time of registration. This certificate can be obtained from any of the regional offices of the Veterans Administration by filing an application and showing discharge papers. Since several weeks may be required in some instances to complete the "Certificate of Eligibility and Entitlement," application should be completed well in advance of registration. An application blank (Form 1950) is enclosed. If time does not permit this procedure to be followed, the veteran may *submit a photostatic copy of his discharge papers* at the time of registration and file his application at that time. In order to verify this photostatic copy, the veteran must also show the original.

The University will notify the Veterans Administration when the veteran is enrolled for instruction. This procedure entitles the veteran to subsistence allowances under the G. I. Bill of Rights, providing he meets

all requirements. Subsistence allowances have recently been increased to $65.00 per month in the case of veterans without dependents and $90.00 per month for veterans with dependents. The University will make no attempt to determine the dependency status of the veteran; this is arranged by the veteran himself.

APPLICATION FORM

Director of Postgraduate Instruction
George Washington University School of Medicine
1335 H Street, N.W., Washington 5, D. C.

Please enroll me for the intensive refresher course in.....
(name of course; dates)
offered by the George Washington University School of Medicine. My check or money order for $25 is enclosed to cover the registration fee. I graduated from the ..... Medical School in .....
(date)
..... I have had .... months formal training and .... months experience in the specialty of ..... I shall purchase the bound volumes if they are offered for sale.

DATE ..... NAME.....
(Please print name and address clear)
STREET ADDRESS.....
CITY AND STATE.....

Note: If it is desired that an attempt be made to secure a room for you, please attach letter indicating your needs.